Response of heart rate to a roller coaster ride

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Br Med J 1989;299:1575

Fairground operators are obliged to display a warning to the effect that people with heart disease should not ride on the roller coasters. A 31 year old man with a history of sustained monomorphic ventricular tachycardia induced by exercise ignored this advice and survived a ride on a roller coaster at the Glasgow Garden Festival without adverse effects. This prompted us to monitor heart rate by ambulatory electrocardiography in normal subjects on the same roller coaster.

Subjects, methods, and results

The study group comprised 13 subjects (seven women and six men) with a mean age of 28 (range 19-36) who did not have cardiac disease detected clinically. They volunteered to undergo ambulatory electrocardiography while on the roller coaster. After abrading the skin lightly we attached electrodes with

Roller coaster at Glasgow Garden Festival

adhesive tape over the second right intercostal space and apex beat to give a modified CS5 lead. The electrocardiograms were recorded on audio cassette tape with an Oxford Medilog 1 recorder and analysed with an Oxford 4500 analyser.

The roller coaster consisted of a double loop corkscrew (called a boomerang), which imparted an acceleration force of 3 g and reached speeds in excess of 64 km/h during forward and reverse runs. The ride lasted 94 seconds.

Twelve good quality electrocardiograms were recorded. No ventricular arrhythmias or episodes of significant ST segment depression were detected. The mean heart rate increased from 69·8 beats/min before the ride to 154·2 (range 130-180) beats/min during it. This represented a mean of 84·5% of the subjects' maximum expected heart rates. All of the subjects reached more than 70% of their maximum expected heart rate, and five reached more than 90% of it. The most striking feature of the electrocardiograms was the speed of onset of the tachycardia. All subjects reached their maximum heart rate in less than eight seconds. The mean time until the heart rate returned to normal was 8·9 (range 2-40) minutes.

Comment

Our results show that a ride on the roller coaster causes a sudden and sustained rise in heart rate and therefore myocardial consumption of oxygen. The heart rate response was similar to that observed during parachute jumping, skiing, and games of squash. This would not be harmful to healthy people but might be hazardous to people with ischaemic heart disease, representing as it does the magnitude of heart rate increase we would expect to induce gradually over several stages of a standard exercise protocol.

Although it was reassuring that no ventricular arrhythmias were induced in these normal subjects, this study confirmed that it is wise to advise patients with cardiac disease not to ride on roller coasters.

We thank the volunteers who took part in the study and Sanofi, United Kingdom, for financial help.

- 1 Jung K, Schulze J. Sports-medical studies on parachute jumpers with particular reference to the behaviour of heart rate. *International Journal of Biotelemetry* and Patient Monitoring 1982;9:238-50.
- 2 Karvonen J, Vuorimaa T. Heart rate and exercise intensity during sports activities. Practical application. Sports Med 1988;5:303-12.
- 3 Northcote RJ, Macfarlane PW, Ballantyne D. Ambulatory electrocardiography in squash players. Br Heart J 1983;50:372-7.
- Bruce RA, Kusumi F, Hosmer D. Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. Am Heart J 1973;85:546-62.

Rat bites and leprosy

Earlier this year, writing about diabetic foot in the West Indies, Dr P Cooles and Mr H Paul noted that people with peripheral neuropathy do not feel rats biting their feet and so the rats can feed undisturbed (BMJ 1989;198:868). In rural Africa leprosy is a more important cause of peripheral neuropathy than diabetes. About 40 years ago, when I was a government medical officer in Nigeria, villagers greatly feared leprosy and drove people with leprosy out of their villages for fear of contagion. Here and there, perhaps where the characteristic clustering of patients occurred, the local authority put up small refuges where they could live and get some sort of care and attention.

One of these centres was a collection of mud huts that was remote from any other habitation and about 150-200 km from my district hospital. I could visit it for only about an hour every four to six weeks. Most of the inmates had advanced leprosy and looked old and wretched; one, however, was a young man in his 20s who

was not yet severely handicapped. Once, made to feel ashamed professionally by their sincere expressions of thanks, I asked if there was anything more that I could do for them. The young man, their spokesman, asked if I could get them a "boos." I did not understand, and he elaborated: "A boosy cat."

- "Have you got mice?" I asked.
- "No sir, rats," he replied.
- "Are they eating your food?"
- "No sir, us."

Then they showed me the linear, parallel toothmarks of rats on the atrophied stumps of their arms and legs.

They got their pussy cat and also better (but still unsatisfactory) arrangements for treatment. It is still one of my most unhappy memories from the 20 years I spent in west Africa.

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